Cytokinetics Announces Positive Phase I Clinical Trial Results for CK-1827452

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South San Francisco, CA, June 29, 2006 - Cytokinetics, Incorporated (Nasdaq: CYTK - News) announced positive results today from a first-in-humans Phase I clinical trial evaluating CK-1827452, a novel cardiac myosin activator, administered intravenously. The clinical trial was designed as a double-blind, randomized, placebo-controlled, dose-escalation trial conducted to investigate the safety, tolerability, pharmacokinetics, and pharmacodynamic profile of a six-hour infusion of CK-1827452 in healthy volunteers.

In this Phase I clinical trial, the maximum tolerated dose (MTD) was determined to be 0.5 mg/kg/hr for the six-hour infusion in healthy volunteers. At this dose, the six-hour infusion of CK-1827452 produced a statistically significant and clinically relevant increase in ejection fraction and fractional shortening as measured from baseline to the end of the infusion in comparison to placebo; these clinically relevant increases in cardiac function were associated with a statistically significant prolongation of systolic ejection time. At the MTD, CK-1827452 was well-tolerated when compared to placebo. Across the dosing levels evaluated in this clinical trial, infusions of CK-1827452 were characterized by linear, dose-proportional pharmacokinetics and produced dose-dependent pharmacodynamic effects.

Data from the Phase I clinical trial of CK-1827452 will be presented at a session entitled "Recent and Late Breaking Trials" at the 10th Annual Meeting of the Heart Failure Society of America on Wednesday, September 13, 2006 in Seattle, Washington. The presentation will be made by John R. Teerlink, M.D., F.A.C.C., F.A.H.A., F.E.C.S., Associate Professor of Medicine at the University of California, San Francisco, and Director of the Heart Failure Clinic, Veterans Affairs Medical Center, San Francisco. Dr. Teerlink is a Co- Principal Investigator and responsible for echocardiographic analysis for the Phase I clinical trial.

The Phase I clinical activity of CK-1827452 announced today is consistent with results from preclinical models which evaluated CK-1827452 in both normal and heart failure dogs. In these models, underlying the increase in ejection fraction and fractional shortening was a dose-related increase in the systolic ejection time, which has now also been observed in humans. Data presented at the 2005 Annual Meeting of the Heart Failure Society of America arising from a dog model of heart failure demonstrated that CK-1827452, administered as a 0.5 mg/kg bolus followed by a 3-4 hour infusion at 0.5 mg/kg/hr, increased cardiac contractility and cardiac output without increasing myocardial oxygen consumption. Preclinical studies have also demonstrated more pronounced effects of CK-1827452 on indices of cardiac function relative to baseline levels in canine models of heart failure in comparison with relative effects achieved in normal dogs.

"We are pleased with the results of this clinical trial. We believe these data provide further validation for the underlying therapeutic hypothesis for CK-1827452, which is being developed as a potentially improved form of inotropic therapy. Its novel mechanism may offer a safer and more effective treatment alternative for patients with compromised cardiac function," stated Andrew A. Wolff, M.D., F.A.C.C., Cytokinetics' Senior Vice President of Clinical Research and Development and Chief Medical Officer. "These results in healthy volunteers nicely recapitulate what we observed in normal dogs and increase our confidence that what we observed in animal models of heart failure may be reproduced in heart failure patients."

"These highly-anticipated data are encouraging for the many patients presently afflicted with heart failure," stated Dr. John Teerlink. "Patients with heart failure have had few new pharmaceutical alternatives for treating their particularly incapacitating and life-threatening disease. Based on the Phase I data, CK-1827452 looks promising as a potential addition to our treatment armentarium for these patients in need."

In the Phase I clinical trial of CK-1827452 announced today, doses that exceeded the MTD of CK-1827452 were associated with longer prolongations of systolic ejection time and larger increases in ejection fraction and fractional shortening than those that were observed with doses at or below the MTD. The corresponding adverse effects at the higher dose levels in humans appear similar to the adverse findings observed in the preclinical safety studies which occurred at similar plasma concentrations. These effects are believed to be related to a hyper-contractile state of the myocardium and were resolved promptly with discontinuation of the infusions of CK-1827452.

Cytokinetics Teleconference Today
Cytokinetics senior management will host a conference call at 10:30 a.m. Eastern Time to discuss summary results of the Phase I clinical trial of CK-1827452. The conference call will be simultaneously webcast and can be accessed in the Investor Relations section of Cytokinetics' website at www.cytokinetics.com. The live audio of the conference call is also accessible via telephone to investors, members of the news media and the general public by dialing either (866) 999-CYTK (2985) (United States and Canada) or (706) 679-3078 (International) and typing in the passcode 2348272.

An archived replay of the webcast will be available via Cytokinetics' website until July 6, 2006. The replay will also be available via telephone from June 29, 2006 at 1:00 p.m. Eastern Time until July 6, 2006 by dialing (800) 642-1687 (United States and Canada) or (706) 645-9291 (International) and typing in the passcode 2348272.

Development Status of CK-1827452
CK-1827452, a novel, small-molecule, direct activator of cardiac myosin, has recently completed a Phase I, first-in-humans clinical trial designed to evaluate this drug candidate as an intravenous formulation in healthy volunteers. At the end of 2005, Cytokinetics also selected CK-1827452 as a potential drug candidate for the treatment of chronic heart failure via oral administration. An oral bioavailability clinical trial of CK-1827452 is planned for the second half of 2006; this clinical trial is designed to inform oral formulation development for CK-1827452. Cytokinetics also expects that CK-1827452 will be entering Phase II clinical trials in the second half of 2006. The international Phase II clinical trials program is planned to evaluate CK-1827452 in a diversity of patients including those with stable heart failure, inducible ischemia, impaired renal function, and acute heart failure. This program is being designed to test the safety and efficacy of CK-1827452, in both intravenous and oral formulations, for the potential treatment of heart failure across the continuum of care, both in the hospitalized setting and in the outpatient setting.

Background on the Heart Failure Market
Heart failure is a widespread and debilitating syndrome affecting approximately five million people in the United States alone. The high and rapidly growing prevalence of heart failure translates into significant hospitalization rates and associated societal costs. The number of hospital discharges in the United States identified with a primary diagnosis of heart failure rose from 550,000 in 1989 to over 1 million in 2003. Heart failure is one of the most common primary discharge diagnoses identified in hospitalized patients over the age 65 in the United States. The annual costs of heart failure in the United States are estimated to be $29.6 billion, including $19.3 billion for inpatient care. According to industry reports, the U.S. market for heart failure drugs was approximately $1.33 billion in 2004. Despite currently available therapies, readmission rates for patients over the age of 65 remain high at

30 to 40 percent within six months of hospital discharge and mortality rates exceed 50% over a five year period following a diagnosis of acute heart failure. The limited effectiveness of current therapies points to the need for next-generation therapeutics that may offer improved efficacy without increased adverse events.

Background on Cardiac Myosin Activators and Cardiac Contractility
Cardiac myosin is the cytoskeletal motor protein in the cardiac muscle cell that is directly responsible for converting chemical energy into the mechanical force resulting in cardiac contraction. Cardiac contractility is driven by the cardiac sarcomere, a highly ordered cytoskeletal structure composed of cardiac myosin, actin and a set of regulatory proteins and is the fundamental unit of muscle contraction in the heart. The sarcomere represents one of the most thoroughly characterized protein machines in human biology. Cytokinetics' cardiovascular program is focused towards the discovery and development of small molecule cardiac myosin activators in order to create next-generation treatments to manage acute and chronic heart failure. Cytokinetics' program is based on the hypothesis that activators of cardiac myosin may address certain mechanistic liabilities of existing positive inotropic agents by increasing cardiac contractility without increasing intracellular calcium, which may be associated with adverse clinical effects in heart failure patients. Current inotropic agents, such as beta-adrenergic receptor agonists or inhibitors of phosphodiesterase activity, increase cardiac cell contractility by increasing the concentration of intracellular calcium, which indirectly activates cardiac myosin; this effect on calcium levels, however, also has been linked to potentially life-threatening side effects. The inotropic mechanism of current drugs also increases the velocity of cardiac contractility and shortens systolic ejection time. In contrast, cardiac myosin activators have been shown to work in the absence of changes in intracellular calcium by a novel mechanism that directly stimulates the activity of the cardiac myosin motor protein. Cardiac myosin activators accelerate the rate-limiting step of the myosin enzymatic cycle and shift the enzymatic cycle in favor of the force producing state. This novel inotropic mechanism results not in an increase in the velocity of cardiac contractility but instead a lengthening of the systolic ejection time resulting in increased cardiac contractility and cardiac output in a potentially more oxygen efficient manner.

About Cytokinetics
Cytokinetics is a biopharmaceutical company focused on the discovery, development and commercialization of novel small molecule drugs that specifically target the cytoskeleton. The cytoskeleton is a complex biological infrastructure that plays a fundamental role within every human cell. Cytokinetics' focus on the cytoskeleton enables it to develop novel and potentially safer and more effective classes of drugs directed at treatments for cancer, cardiovascular disease and other diseases. Under a strategic alliance established in 2001, Cytokinetics and GlaxoSmithKline are collaborating to develop and commercialize small molecule therapeutics targeting human mitotic kinesins for applications in the treatment of cancer and other diseases. Ispinesib (SB-715992), SB-743921 and GSK-923295 are being developed under the strategic alliance with GSK. GSK is conducting Phase II and Ib clinical trials for ispinesib and a Phase I clinical trial for SB- 743921, and Cytokinetics is conducting a Phase I/II trial of SB-743921 in non-Hodgkin's lymphoma. Cytokinetics' unpartnered cardiovascular disease program is the second program to leverage the company's expertise in cytoskeletal pharmacology. Cytokinetics recently completed a Phase I clinical trial with CK-1827452, a novel small molecule cardiac myosin activator, for the intravenous treatment of heart failure and also is advancing CK-1827452 as a potential drug candidate for the treatment of chronic heart failure via oral administration. Additional information about Cytokinetics can be obtained at http://www.cytokinetics.com.

This press release contains forward-looking statements for purposes of the Private Securities Litigation Reform Act of 1995 (the "Act"). Cytokinetics disclaims any intent or obligation to update these forward-looking statements, and claims the protection of the Safe Harbor for forward-looking statements contained in the Act. Examples of such statements include, but are not limited to, statements about the timing, scope and focus of our clinical research and development activities with respect to CK-1827452, the size and growth of expected markets for CK-1827452, the potential benefits of our drug candidates and potential drug candidates, and the benefits of data obtained from completed clinical trials. Such statements are based on management's current expectations, but actual results may differ materially due to various factors. Such statements involve risks and uncertainties, including, but not limited to, those risks and uncertainties relating to difficulties or delays in patient enrollment for clinical trials, unexpected adverse side effects or inadequate therapeutic efficacy of our drug candidates and other potential difficulties or delays in development, testing, regulatory approval, production and marketing of Cytokinetics' drug candidates that could slow or prevent clinical development, product approval or market acceptance (including the risks relating to uncertainty of patent protection for Cytokinetics' intellectual property or trade secrets, Cytokinetics' ability to obtain additional financing if necessary and unanticipated research and development and other costs), changing standards of care and the introduction of products by competitors or alternative therapies for the treatment of indications currently or potentially targeted by CK-1827452 and the implementation and maintenance of procedures, policies, resources and infrastructure relating to compliance with new or changing laws, regulations and practices. For further information regarding these and other risks related to Cytokinetics' business, investors should consult Cytokinetics' filings with the Securities and Exchange Commission.